3. The Transition Stage from Midbrain Syndrome to Traumatic Apallic Syndrome

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In those cases of apallic syndrome which occur after acute brain injury, an acute midbrain syndrome is observed as a preliminary stage. This syndrome, caused by tentorial strangulation, develops into the complete stage in three phases. It is characterized clinically by increased curtailment of consciousness to a comatose state, release of the motor functions of the midbrain with increased stretching of the extremities and trunk, and finally, by accompanying stretch synergisms in the complete state of the syndrome, as well as a release of the vegetative systems of the midbrain (with hyperpnea, trachycardia, hypertonia, and hyperthermia (Gerstenbrand, 1967). If intracranial pressure persists, a strangulation in the foramen occipitale magnum can develop and thereby an acute syndrome of the medulla oblongata (bulbar brain syndrome) can occur, which develops after subsidence of the symptoms of midbrain damage (McNeal and Plum, 1962; Müller, 1965). When the strangulation pressure decreases, a development from the bulbar brain syndrome to the midbrain syndrome is possible only if the bulbar brain symptoms have not existed longer than about 30 min. The acute midbrain syndrome can revert back through the same phases which are to be observed in the development of the complete state of the disease. The acute bulbar brain syndrome, however, can also develop into a irreversible loss of brain function, a condition which corresponds clinically to the death of the brain.

With patients who show an apallic syndrome after acute injury to the cerebrum following hypoxia or diffuse encephalitis, the phaselike development to a acute midbrain syndrome is generally not evident, nor is the complete state of the disease well defined. The same holds true for the apallic syndrome patients with exogenous or endogenous poisoning, such as mercury or insulin poisoning. In these cases the otherwise clearly defined acute midbrain symptoms are changed by local injury in the region of the brain stem.

If the acute midbrain symptoms do not decrease directly, a subsidence of the symptoms sets in after a few days, and the complete stage of an apallic syndrome develops after 10-20 days. This phase—the observation of which is of great importance for the prognosis of acute midbrain syndrome in serious brain injury—is termed the "transition stage" (Gerstenbrand, 1967). Individual phases of the transition stage are derived from the chief symptom, disturbance of consciousness. The further symptom categories, such as the release of the motor system and vegetative functions, can be assigned to the characteristic state of consciousness in each case. The development of the transition stage to the apallic syndrome can best be exemplified by discussing the recovery after a secondary acute midbrain syndrome following head injury. Corresponding to the disturbance of consciousness existing in the transition stage, we have termed the three definable stages as follows: (1) *coma prolongé* (Fau, 1956; Vigouroux et al., 1964), (2) *parasomnia* (Jefferson, 1944), and

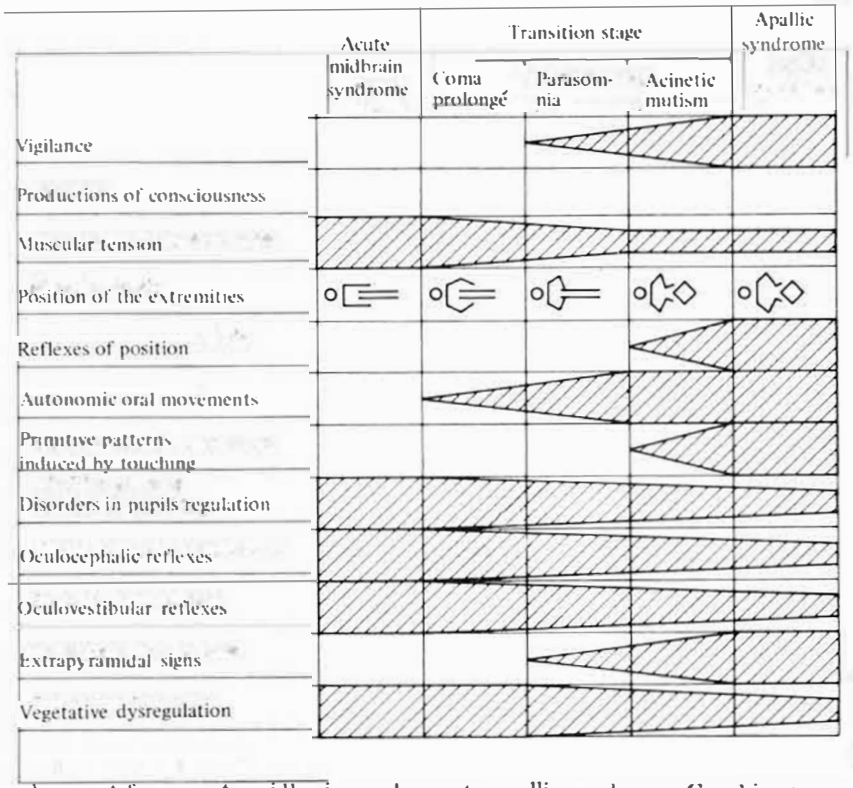

Fig. 1. Development from acute midbrain syndrome to apallic syndrome. Graphic representation of cardinal symptoms

1. Coma Prolongé

Although deep unconsciousness continues (*coma prolongé*) and the patient shows no reaction to external stimuli, the spontaneous stretching spasms subside generally after 3-5 days, and in this phase are only actuated by pain stimuli, initially in pronounced form, but later to a lesser degree. The extremities are stretched out and the patient's trunk and neck are also extended. At the end of this stage, however, a flexed position of the arms begins to set in, which can then be intensified by stimulation. The heightened tension of the body muscular system is considerably increased; coordinated movements of any kind are lacking. The tendon reflexes are increased; Babinski reflexes are generally more easily actuated than in the acute midbrain syndrome.

After the Babinski examination, an increased tendency to stretch the extremities is sometimes observed. The eyeballs are divergent but no longer in a fixed position. The oculocephalic reflexes are only slightly actuated; the vestibuloocular reflex shows an initial reaction to heightened tension. The reaction to light of the somewhat less than medium-wide

In the course of this stage spontaneous chewing mechanisms set in. At the beginning, these exist only in a few chewing movements, whereby the lips are slightly open, but sometimes they remain closed too, so that the movements are carried out only by the lower jaw. Poeck and Hubach (1963) interpret these movement lapses as a primitive type of oral mechanism—not one connected with the environment, but an organized one nonetheless.

This stage can last 3-7 days, before changing into the "parasomnia" stage.

2. Parasomnia

The patient continues to be unconscious, lies with closed eyes, and shows no reaction to external stimuli. To intensive pain stimuli, however, the mass movements of the upper extremities already mentioned set in with a simultaneous increase in the stretching of the legs. The coma seems to have become superficial and resembles a state similar to sleep (parasomnia).

The spontaneous stretching of the extremities changes, in an intensified manner, into the flexed position which can be particularly seen in the upper extremities. A heightened tendency toward flexion is produced by pain stimuli. With severe pain stimuli an extension of the upper extremities can set in, following the above-mentioned tendencies to mass movements of the arms.

The heightened tension of the muscular system is still evident, but decreases further in its intensity. At this stage there may be a deviation of the eyeballs and the head, accompanied by the holding of the asymmetrically hypertensive neck reflex. The tendon reflexes are enhanced; the pyramidal signs are actuated. The divergence of the eyeballs has further diminished and remains variable; the eyeballs roll slightly. The oculocephalic reflex is actuated; the vestibuloocular reflex shows a hypertensive reaction. The vegetative reactions demonstrate a further stabilization; the sympathetic reaction state is not so strongly pronounced; with pain stimuli, however, an emergency reaction is produced. The automatic chewing actions appear at this stage in augmented form; they are spontaneous or actuated by external stimuli, such as the introduction of a nasal probe, etc. At the end of this stage the lip-closing and snout reflex can be demonstrated, and the bulldog reflex is slightly present. Hypertensive clutching is present in a number of cases. The parasomnia stage can also last 3-5 days.

3. Akinetic Mutism

At this stage patients open their eyes, initially for short periods of time, later for more extended periods. However, the blinking and menacing reflexes are not present.

During those periods of time when the patient has his eyes open, an increased tendency to vegetative stimulis response exists. The vegetative hypertension is shifted to the sympathetic tense state. In the following periods of a condition resembling sleep, a parasympathetic state of hypertension predominates. In his posture the patient shows a slight flexing tendency; also present in the legs, while the arms demonstrate a clear bending posture. The pronounced

mentioned tendency to asymmetric, hypertensive neck reflex can increase. After pain stimuli, mass movements of the arms and, to a lesser degree, also of the legs, latently appear. With intensive pain stimuli, an increase in the flexing and stretching posture of the extremities follows. The muscle reflexes are intensified, the hypertension of the muscular system is heightened, and the pyramidal signs can be actuated, resulting occasionally in a flexing movement and a flight reflex in the legs. The eyeballs of the patient are in a position of divergence, spontaneous horizontal movements occur. The oculocephalic reflexes are actuated, the vestibuloocular reflex is heightened tonically. The medium-wide pupils show a lively reaction to light, the ciliospinal reflex is clearly evident. The vegetative functions have been stabilized but still remain displaced in the sympathetic tonic condition.

Primitive motor patterns show a further differentiation. Tactile oral adjustment mechanisms are carried out which are generally followed by spontaneous automatic chewing movements. Lip-closing and snout reflexes, as well as the bulldog reflex, are clearly apparent at this stage. The asymmetric tonic neck reflex is generally evident. Sporadically Babkin reflex can be actuated; seldom can a Chotzko reflex be demonstrated. The mental reflexes and the glabellar reflexes are present in almost all cases.

The third phase of the transition stage is in most cases curtailed compared with the other two, and lasts 2-5 days. It follows the overall picture of the apallic syndrome.

Compared with this phaselike development in cases with a traumatic apallic syndrome or after tentorial strangulation from a supratentorial increase in volume of different etiology, the symptoms in the transition stage after hypoxic damage and also after poisonings are generally less well definable. This can be explained particularly by the intruding symptoms conditioned by the brain stem.

In the time lapse of the transition stage, prognostic inferences can be made from various long pauses of the individual intermediary phases. A pause in the first two sections, over a period of 5 days each, is to be interpreted prognostically as an unfavorable sign; generally a temporal extension of the *coma prolongé* stage over 7 days appears particularly unfavorable. Thus the intensification of hypertension and/or the presence of permanent stretch synergisms remains the most important factor, together with the disturbance of consciousness, for an unfavorable prognosis.

With exact registration of the development of symptoms in the transition stage, an orientation in the sphere of transition between the acute midbrain syndrome and the apallic syndrome is possible.

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